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Bacteria associated with clinical postpartum dysgalactia syndrome in farmed sows in the Republic of Macedonia

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Abstract: The objective of this study was to gather information about the prevalence of bacterial species in farmed sows, with special focus on the clinical manifestation of postpartum dysgalactia syndrome (PDS). One hundred and sixteen sows from 5 pig farms in the Republic of Macedonia were clinically examined for PDS 12–24 h after farrowing. Milk samples and vaginal swabs for bacteriological testing were taken from PDS-affected (PDSA, n = 30) and PDS-unaffected (PDSU, n = 86) sows. *Escherichia coli*, staphylococci, and streptococci were the predominant bacteria isolates. *Escherichia coli* was the most frequently found isolate, with a prevalence of 73.3% in PDSA and 31.4% in PDSU sows. Compared to PDSU sows, *Escherichia coli* was more prevalent in both milk (53.8% vs. 31.4%) and vaginal swabs (74.3% vs. 47.1%) from PDSA sows. Greater prevalence of *Escherichia coli* in vaginal swabs (66.7%) from PDSA sows suggests that the genital tract represents a possible route for transmission of the infection and that *Escherichia coli* plays a major role in the development of clinical PDS. Further investigation should be made in order to identify whether specific virulent factors of this bacterium isolated from the genital tract of PDSA sows are associated with clinical occurrence of the syndrome.

Key words: Bacteria, postpartum dysgalactia syndrome, sow

1. Introduction

Sufficient colostrum and milk production in the first days after farrowing are essential for the survival and growth of piglets. Every disturbance in a sow's milk production leads to decreased weight gain and increased mortality rate in newborn piglets (1,2). Postpartum dysgalactia syndrome (PDS) in sows is characterized by fever and reduced milk production in the first 12 to 48 h postpartum (1,3,4). The most often used term in older literature, mastitis, metritis, and agalactia syndrome, nowadays is considered as a subclass of PDS, since in many cases there is no true agalactia (5). It is an important disease complex associated with major economic losses and animal welfare issues in pig production worldwide (6,7). The incidence of this pathological condition at herd level is estimated to vary between 0.5% and 60% (8), with an average of 13% (7,9,10,11). There are numerous clinical signs of PDS that vary from herd to herd, but the most frequent signs seen in sows are fever, metritis, mastitis, hypogalactia/agalactia, anorexia, cystitis, constipation, and depression (7,8,12,13). Besides the multifactorial nature of PDS, it is considered that bacteria play a major role in its etiology (6). Although bacterial invasion of the mammary gland via the teat canal is currently the major route of infection, urinary and genital infections are also considered to be a major source for subsequent infection of the uterus and the mammary glands (8,14). It is generally accepted that coliform bacteria from the genera *Escherichia*, *Enterobacter*, *Citrobacter*, and *Klebsiella* are the most important pathogens strongly related to PDS in sows (1,15–17). There is a lack of data about the prevalence of bacteria associated with PDS in farmed sows in the Republic of Macedonia. The objective of the present study was to determine the prevalence and species of bacteria in vaginal swabs and milk samples from farmed sows with special focus on the postpartum clinical status of the sows.

2. Materials and methods

One hundred and sixteen sows from five commercial pig farms in Macedonia were included in this study. The sows were of different parity (1–9) and different genetic lines (Landrace-Yorkshire F1 and Dalland hybrid). All sows were clinically examined for the presence of PDS 12–24 h after farrowing based on predetermined clinical signs (Table 1).

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Table 1. Prevalence of clinical signs found in PDSA sows.

Clinical sign	Description	PDSA sows %
Fever	Increased rectal temperature (≥39.5 °C)	46.7 (14/30)
Pathological vulvar discharge	Copious purulent vulvar discharge	73.3 (22/30)
Mastitis	Warm, painful, swollen, and firm mammary glands	66.7 (20/30)
Reduced appetite	Less than half of the quantity of feed provided	36.7 (11/30)
Hypogalactia	Reduced milk flow (drops of milk)	70 (21/30)
Depression	Lethargy and sternal recumbency	56.7 (17/30)
Altered piglet behavior	Lethargy, restlessness, vigorous nursing efforts	60 (18/30)

The sows were classified as PDS-affected if at least two clinical signs were found.

After clinical assessment, vaginal swabs and milk samples for bacteriological testing were taken from PDS-affected (PDSA, n = 30) and PDS-unaffected (PDSU, n = 86) sows. In total, 232 samples (116 vaginal swabs and 116 milk samples) were collected. Prior to milk sampling, the mammary glands were cleaned and disinfected with 70% ethanol. Pooled milk samples from at least three mammary glands were collected into sterile plastic cups 5 min after oxytocin injection (30 I.E., i.m.). The first stream was discarded and the second and third were milked into the sterile cups. Before taking vaginal swabs, the vulva was cleaned and disinfected with 10% iodine solution. Vaginal swabs were taken using sterile metal speculums, deeply inserted into the vagina and by thorough contact with the ventral mucosa for at least 10 s. Both vaginal swabs and milk samples were stored at 4 °C and transported to the laboratory within 2 h.

Bacteriological testing was performed by using routine diagnostic procedures. The initial inoculation of the samples was performed on 5% sheep blood agar (blood agar base (Merck, Germany)] and selective media for gram-negative bacteria [(Xylose Lysine Deoxycholate, MacConkey, and Tryptone Bile X-Glucuronide Agar (Merck, Germany)]. After 24 h of aerobic incubation at 37 °C, the grown bacteria were distinguished by their morphology, hemolysis on blood agar catalase reaction, Gram staining, and growth on selective media. Selected colonies were subcultivated on blood agar for another 24 h at 37 °C in order to obtain pure cultures. The final identification was performed by automated system VITEK 2 Compact (BioMérieux, France).

To determine the statistical differences in bacterial prevalence between samples from PDSA and PDSU sows, we performed the chi square-test and the results were considered statistically significant at $P < 0.05$.

3. Results

In total, 88/116 (75.9%) of sows were positive on bacteriological testing, and 136 bacterial isolates were recovered. From PDSA sows, 48 isolates were proven in vaginal swabs (35 isolates) and milk samples (13 isolates), while 88 isolates were found in vaginal swabs (53 isolates) and milk samples (35 isolates) from PDSU sows. Most of the isolated bacteria belonged to the families *Enterobacteriaceae*, *Staphylococcaceae*, and *Streptococcaceae*, but *Escherichia coli* was the most frequent isolate with a prevalence of 73.3% in PDSA and 31.4% in PDSU sows (Table 2). Regarding the presence of bacterial isolates in the different samples (vaginal and milk), *E. coli* was the most dominant bacterium found in both PDSA and PDSU sows (Table 3). The bacteria from the genus *Staphylococcus* were more frequently found in the samples from PDSU sows (Table 2), and the most dominant isolate was *Staphylococcus hyicus* (Table 3). On the other hand, streptococci were more present in the vaginal swabs from PDSA sows (Table 4), where *Aerococcus viridians* was the most isolated species (Table 3). Compared to PDSU sows, *E. coli* was detected in significantly higher percentages in vaginal swabs and milk samples from PDSA sows (Table 4). Additionally, *E. coli* in PDSA sows was also more predominantly found in vaginal swabs (66.7%) than in milk (23.3%). A highly significant difference was observed in the number of vaginal swabs with no bacteriological growth from PDSU sows in comparison to the vaginal swabs from PDSA sows (Table 4).

4. Discussion

The present study indicates a significant difference in the prevalence of bacteria associated with clinical manifestation of PDS in sows. In general, the most important and one of the most common clinical signs used for diagnosis of PDS in sows is increased rectal temperature (≥39.5 °C) 12–48 h postpartum (1,18). However, increased

Table 2. Prevalence of bacteria species in all tested sows.

Bacteria species	PDSA sows %	PDSU sows %
Escherichia coli	73.3 (22/30)	31.4 (27/86)
Staphylococcus spp.	10 (3/10)	20.9 (18/86)
Streptococcus spp.	10 (3/10)	9.3 (8/30)
Staphylococcus hyicus	0.0 (0/30)	7.0 (6/86)
Sphingomonas paucimobilis	0.0 (0/30)	7.0 (6/86)
Aerococcus viridians	10 (3/10)	5.8 (5/86)
Staphylococcus simulans	0.0 (0/30)	2.3 (2/86)
Sphingobacterium talpophilum	3.3 (1/30)	1.2 (1/86)
Staphylococcus chromogenes	0.0 (0/30)	2.3 (2/86)
Staphylococcus. haemoliticus	3.3 (1/30)	1.2 (1/86)
Streptococcus suis 1	3.3 (1/30)	0.0 (0/86)
Streptococcus pseudoporcinus	3.3 (1/30)	0.0 (0/86)
Streptococcus sanguinis	3.3 (1/30)	0.0 (0/86)
Klebsiella pneumonia	0.0 (0/30)	1.2 (1/86)
Yersinia enterocolitica	0.0 (0/30)	1.2 (1/86)
Pasteurella spp.	0.0 (0/30)	1.2 (1/86)
Pasteurella pneumotropica	0.0 (0/30)	1.2 (1/86)
Acinetobacter lwoffii	0.0 (0/30)	1.2 (1/86)
Kocuria varians	0.0 (0/30)	1.2 (1/86)
Cedecea davisae	0.0 (0/30)	1.2 (1/86)
Leuconostoc mesenteroides subsp. *cremoris*	3.3 (1/30)	0.0 (0/86)

rectal temperature sometimes leads to misdiagnosis, since physiological hyperthermia is often observed in sows in the first 24 h postpartum (4). Besides rectal temperature, other clinical signs such as mastitis, pathological vulvar discharge, reduced appetite, and altered piglet behavior are also relevant for PDS diagnosis (8). In our study, we have diagnosed the syndrome by performing detailed clinical examination of the selected sows 12–24 h after farrowing, as suggested by other authors (4,8,14). Pathological vulvar discharge was the most common clinical sign that we observed in PDSA sows, which is in accordance with the study by Madec and Leon (9), where 64.5% of the sows suffered puerperal disease exhibiting vulvar discharge.

The most frequent bacteria that we isolated in all sows were *E. coli*, staphylococci, and streptococci. This bacterial spectrum is in agreement with other studies (4,19). Kemper and Gerjets (4) found that the most frequent bacteria in PDSA and PDSU sows were representatives of the families *Enterobacteriaceae*, *Staphylococcaceae*, *Streptococcaceae*, and *Enterococcaceae*. In our study, we found that *E. coli* was the most prevalent bacterium in both milk samples and vaginal swabs from PDSA sows, which is in agreement with previous studies (8,15,20). In vaginal swabs and milk samples from 187 sows showing clinical signs of PDS, Hirsch et al. (8) detected mainly *E. coli*, but also *Staphylococcus* spp., *Streptococcus* spp., and *Enterococcus* spp. In the study of Wegmann et al. (15), from 131 mammary complexes of PDSA sows, *E. coli* and *Klebsiella pneumoniae* were the most frequent bacteria isolated. However, in our study, *E. coli* in PDSA sows was less frequently found in milk than in vaginal swabs (Table 4). This higher prevalence of *E. coli* in the vaginal swabs of PDSA sows conforms to the findings of other researchers (14,21,22). Bostedt et al. (21) documented that *E. coli* was the predominant bacterium in the genital tract of 78 gilts suffering from puerperal septicemia, and Waller et al. (22) reported that this bacterium was the most frequent one found in urine and vulvar discharges of periparturient diseased sows. On the other hand, our results regarding the prevalence of gram-negative bacteria in the genital tract of PDSA sows disagrees with the findings of other authors. Armstrong et al. (23), Nachreiner and Ginther (24), and Morkoc et al. (25) supported the theory that the uterus has a minor role in the etiology of PDS, since gram-negative bacteria were less frequently isolated from this organ. The importance of *E. coli* in the etiology of PDS

Table 3. Number of bacterial isolates in the samples from all tested sows.

Bacterial species		PDSA sows		PDSU sows	
	n	Milk	Vaginal	Milk	Vaginal
Escherichia coli	69 (50.7%)	7 /13 (53.8%)	26/35 (74.3%)	11/35 (31.4%)	25/53 (47.1%)
Staphylococcus spp.**	21 (15.4%)	1/13 (7.7%)	2/35 (5.7%)	5/35 (14.3%)	13/53 (24.5%)
Streptococcus spp.**	10 (7.4%)	0/13 (0.0%)	3/35 (8.6%)	5/35 (14.3%)	2/53 (3.8%)
Staphylococcus hyicus	8 (5.9%)	0/13 (0.0%)	0/35 (0.0%)	6/35 (17.1%)	2/53 (3.8%)
Sphingomonas paucimobilis	7 (5.1%)	0/13 (0.0%)	0/35 (0.0%)	1/35 (2.8%)	6/53 (11.3%)
Aerococcus viridians	4 (2.9%)	1/13 (7.7%)	2/35 (5.7%)	1/35 (2.8%)	0/53 (0.0%)
Staphylococcus simulans	2 (1.5%)	0/13 (0.0%)	0/35 (0.0%)	2/35 (5.7%)	0/53 (0.0%)
Sphingobacterium talpophilum	2 (1.5%)	0/13 (0.0%)	1/35 (2.9%)	0/35 (0.0%)	1/53 (1.9%)
Staphylococcus chromogenes	1 (0.7%)	0/13 (0.0%)	0/35 (0.0%)	1/35 (2.8%)	1/53 (1.9%)
Staphylococcus. haemoliticus	1 (0.7%)	1/13 (7.7%)	0/35 (0.0%)	0/35 (0.0%)	0/53 (0.0%)
Streptococcus suis 1	1 (0.7%)	1/13 (7.7%)	0/35 (0.0%)	0/35 (0.0%)	0/53 (0.0%)
Streptococcus pseudoporcinus	1 (0.7%)	0/13 (0.0%)	1/35 (2.9%)	0/35 (0.0%)	0/53 (0.0%)
Streptococcus sanguinis	1 (0.7%)	1/13 (7.7%)	0/35 (0.0%)	0/35 (0.0%)	0/53 (0.0%)
Klebsiella pneumonia	1 (0.7%)	0/13 (0.0%)	0/35 (0.0%)	1/35 (2.8%)	0/53 (0.0%)
Yersinia enterocolitica	1 (0.7%)	0/13 (0.0%)	0/35 (0.0%)	0/35 (0.0%)	0/53 (0.0%)
Pasteurella spp.	1 (0.7%)	0/13 (0.0%)	0/35 (0.0%)	0/35 (0.0%)	1/53 (1.9%)
Pasteurella pneumotropica	1 (0.7%)	0/13 (0.0%)	0/35 (0.0%)	1/35 (2.8%)	0/53 (0.0%)
Acinetobacter lwoffii	1 (0.7%)	0/13 (0.0%)	0/35 (0.0%)	0/35 (0.0%)	1/53 (1.9%)
Kocuria varians	1 (0.7%)	0/13 (0.0%)	0/35 (0.0%)	0/35 (0.0%)	1/53 (1.9%)
Cedecea davisae	1 (0.7%)	0/13 (0.0%)	0/35 (0.0%)	1/35 (2.8%)	0/53 (0.0%)
Leuconostoc mesenteroides subsp. *cremoris*	1 (0.7%)	1/13 (7.7%)	0/35 (0.0%)	0/35 (0.0%)	0/53 (0.0%)
Total	136 (100%)	13 (9.6%)	35 (25.7%)	35 (25.7%)	53 (39%)

** Not further identified.

Table 4. Statistical associations between the prevalence of most frequently isolated bacteria in different samples.

Bacteria species	Vaginal swabs		P-values	Milk samples		P-values
	PDSA %	PDSU %		PDSA %	PDSU %	
Escherichia coli	66.7 (20/30)	29.1 (25/86)	<0.001	23.3 (7/30)	9.3 (8/86)	<0.05
Staphylococcus spp.	6.7 (2/30)	17.4 (15/86)	<0.05	6.7 (2/30)	16.3 (14/86)	0.05
Streptococcus spp.	16.7 (5/30)	3.5 (3/86)	<0.05	10 (3/30)	7 (6/86)	0.06
No bacterial growth	10 (3/30)	43 (37/86)	<0.001	60 (18/30)	68.6 (59/86)	0.18

has been emphasized and confirmed in several studies (16,17,26–28). Magnusson et al. (17) induced coliform mastitis in sows by intramammary inoculation of *E. coli*, causing clinical and hematological changes similar to natural infection. Furthermore, lipopolysaccharide (LPS) endotoxins produced by gram-negative bacteria, especially those from *E. coli*, are strongly related with the pathogenesis of PDS (16). These endotoxins administrated via intramammary, intravenous, intrauterine, or subcutaneous routes can induce a series of complex reactions in sow (1). For instance, intramammary LPS infusion (LPS-*Escherichia coli* 0111:B4) leads to elevation of blood cortisol concentrations and proinflammatory cytokines (26). Endotoxins also suppress the release

of prolactin by the pituitary gland, increasing cortisol concentration and decreasing circulating thyroid hormone concentrations (27). All these changes negatively affect the milk production in lactating sows.

Bacteria involved in the etiology of PDS may enter via the mammary gland, uterus, urinary bladder, and the gut (13). The main route of infection is the galactogenous route via the teat canal, which was hypothetically supported by the experimental work of Bertschinger et al. (20), where coliform mastitis was less often found in sows when their mammary glands were protected from fecal contamination. In general, urine and feces excreted by the sow are the main sources of coliform bacteria associated with PDS (1,14). Awad Masalmeh et al. (19) confirmed the fecal origin of *E. coli* and found identical O-serogroups in both fecal and milk samples of PDSA sows. Furthermore, Waller et al. (22) revealed that *E. coli* was the most common organism found in sows with bacteriuria. Therefore, urinary excretion of bacteria has to be considered as a possible source of infection. Additionally, urinary tract infections are strongly related with the infections of the uterus and mammary glands, whereas ascending bacterial invasion via mucosal surfaces, blood vessels, and lymphatics from the infected urinary bladder is possible during the periparturient period (14,23). Although urinary infections caused by *E. coli* are usually associated with puerperal diseases in sows, sometimes clinical signs are missing (22,29). Thus, the high prevalence of *E. coli* in the genital tract of diseased sows obtained in our research may be related with already existent subclinical urinary infections. Additionally, the many pathological vulvar discharges that we observed in PDSA sows indicate a strong connection with PDS. In this context, urinary infection and vulvar discharge predispose to PDS, since decomposed uterine tissue represents an excellent medium for the growth of various opportunistic bacteria (14). Furthermore, the sow's reproductive tract is subjected to infection immediately after farrowing, because of increased numbers of nonpathogenic and facultative pathogenic bacteria present in both the bladder and caudal vagina (14). Besides the infectious factor, there are other risk factors strongly associated with the clinical manifestation of PDS, such as nutrition, housing, microclimate conditions, management, and especially the hygiene practices (6). Therefore, clinical PDS is mainly dependent on the individual predisposition of the sow to combat potential pathogens and the influence of the aforementioned risk factors.

In summary, our results show that the most prevalent bacterium strongly associated with the clinical manifestation of PDS in affected sows is *E. coli*. The higher percentage of *E. coli* found in the vaginal swabs in contrast to milk samples of PDSA sows indicates a possible essential role of the genital tract in the etiology and clinical manifestation of the syndrome. Therefore, further research is needed to identify whether the presence of specific virulence genes of *E. coli* isolated from vaginal swabs in affected sows is associated with the clinical appearance of PDS.

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